d his

(FILE 'HOME' ENTERED AT 11:06:15 ON 21 MAR 2005)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT 11:07:06 ON 21 MAR 2005

L1 2868 S (LIGHT CHAIN VARIAB?)
L2 1348 S L1 AND ANTIGEN?
L3 11 S L2 AND MULTIVALEN?
L4 11 DUPLICATE REMOVE L3 (0 DUPLICATES REMOVED)
L5 2 S L1 AND (HYDROPHOBIC RESIDUE)
L6 2 DUPLICATE REMOVE L5 (0 DUPLICATES REMOVED)
L7 510 S ANTIBOD? AND (HYDROPHOBIC RESIDUE)
L8 185 DUPLICATE REMOVE L7 (325 DUPLICATES REMOVED)
L9 105 S L8 AND BIND?
L10 7 S L9 AND CDR?
L11 7 S L9 AND VARIABL?
L12 3 S L11 NOT L10

d his

(FILE 'HOME' ENTERED AT 11:06:15 ON 21 MAR 2005)

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L1 2868 S (LIGHT CHAIN VARIAB?)
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L7 510 S ANTIBOD? AND (HYDROPHOBIC RESIDUE)
L8 185 DUPLICATE REMOVE L7 (325 DUPLICATES REMOVED)
L9 105 S L8 AND BIND?
L10 7 S L9 AND CDR?
L11 7 S L9 AND VARIABL?
L12 3 S L11 NOT L10

ANSWER 3 OF 3 MEDLINE on STN

AN 80145725 MEDLINE

DN PubMed ID: 6767243

TI Self-association of human immunoglobulin kappa I light chains: role of the third hypervariable region.

AU Stevens F J; Westholm F A; Solomon A; Schiffer M

SO Proceedings of the National Academy of Sciences of the United States of America, (1980 Feb) 77 (2) 1144-8.
Journal code: 7505876. ISSN: 0027-8424.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198005

ED Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19800514

AB Gel electrophoresis and molecular sieve chromatography were used to compare 17 different human kappa I type Bence Jones proteins including 5 for which the amino acid sequence is known. Although electrophoresis in the presence of NaDodSO4 showed uniformity of covalent dimer and monomer molecular weights, Sephadex chromatography under nondissociating conditions showed that monomers eluted with different apparent molecular weights. These differences were attributed to heterogeneity in light chain self-association; dimerization constants of the 17 proteins, calculated from a computer simulation of their behavior upon gel filtration, ranged from less than 10(3) to greater than 10(6) M-1. The **variable** region, more specifically the third hypervariable region, appears to be responsible for the variation in the dimerization constant. Association properties of light chains of known sequence suggest that the presence of an aromatic or **hydrophobic residue** at position 96 enhances dimer formation whereas a charged residue at that position results in light chains remaining stable monomers. The location of hypervariable residue 96 within the amino-terminal portion of the joining segment of the **variable** region suggests that the joining region may account for the variability of self-association of light chains and, moreover, that it has a function in determining the selective association of immunoglobulin polypeptide chains.

CT Amino Acid Sequence
***Binding Sites, Antibody**
Chromatography, Gel
Electrophoresis, Polyacrylamide Gel
Humans
***Immunoglobulin Variable Region**
*Immunoglobulins, Light-Chain
Immunoglobulins, Light-Chain: IM, immunology
*Immunoglobulins, kappa-Chain
Immunoglobulins, kappa-Chain: IM, immunology
Macromolecular Substances
Protein Binding
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.

CN 0 (**Binding** Sites, **Antibody**); 0 (Immunoglobulin **Variable** Region); 0 (Immunoglobulins, Light-Chain); 0 (Immunoglobulins, kappa-Chain); 0 (Macromolecular Substances)

WO 9413806
multivalent single chain antibodies

WO 9738102
US 6,239,259

? 5,877,291

6,492,123 (date)

ANSWER 3 OF 3 MEDLINE on STN

AN 80145725 MEDLINE

DN PubMed ID: 6767243

TI Self-association of human immunoglobulin kappa I light chains: role of the third hypervariable region.

AU Stevens F J; Westholm F A; Solomon A; Schiffer M

SO Proceedings of the National Academy of Sciences of the United States of America, (1980 Feb) 77 (2) 1144-8.
Journal code: 7505876. ISSN: 0027-8424.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198005

ED Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19800514

AB Gel electrophoresis and molecular sieve chromatography were used to compare 17 different human kappa I type Bence Jones proteins including 5 for which the amino acid sequence is known. Although electrophoresis in the presence of NaDodSO4 showed uniformity of covalent dimer and monomer molecular weights, Sephadex chromatography under nondissociating conditions showed that monomers eluted with different apparent molecular weights. These differences were attributed to heterogeneity in light chain self-association; dimerization constants of the 17 proteins, calculated from a computer simulation of their behavior upon gel filtration, ranged from less than 10(3) to greater than 10(6) M-1. The **variable** region, more specifically the third hypervariable region, appears to be responsible for the variation in the dimerization constant. Association properties of light chains of known sequence suggest that the presence of an aromatic or **hydrophobic residue** at position 96 enhances dimer formation whereas a charged residue at that position results in light chains remaining stable monomers. The location of hypervariable residue 96 within the amino-terminal portion of the joining segment of the **variable** region suggests that the joining region may account for the variability of self-association of light chains and, moreover, that it has a function in determining the selective association of immunoglobulin polypeptide chains.

CT Amino Acid Sequence
***Binding Sites, Antibody**
Chromatography, Gel
Electrophoresis, Polyacrylamide Gel
Humans
***Immunoglobulin Variable Region**
*Immunoglobulins, Light-Chain
Immunoglobulins, Light-Chain: IM, immunology
*Immunoglobulins, kappa-Chain
Immunoglobulins, kappa-Chain: IM, immunology
Macromolecular Substances
Protein Binding
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.

CN 0 (**Binding** Sites, **Antibody**); 0 (Immunoglobulin **Variable** Region); 0 (Immunoglobulins, Light-Chain); 0 (Immunoglobulins, kappa-Chain); 0 (Macromolecular Substances)

ANSWER 3 OF 3 MEDLINE on STN

AN 80145725 MEDLINE

DN PubMed ID: 6767243

TI Self-association of human immunoglobulin kappa I light chains: role of the third hypervariable region.

AU Stevens F J; Westholm F A; Solomon A; Schiffer M

SO Proceedings of the National Academy of Sciences of the United States of America, (1980 Feb) 77 (2) 1144-8.
Journal code: 7505876. ISSN: 0027-8424.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198005

ED Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19800514

AB Gel electrophoresis and molecular sieve chromatography were used to compare 17 different human kappa I type Bence Jones proteins including 5 for which the amino acid sequence is known. Although electrophoresis in the presence of NaDodSO4 showed uniformity of covalent dimer and monomer molecular weights, Sephadex chromatography under nondissociating conditions showed that monomers eluted with different apparent molecular weights. These differences were attributed to heterogeneity in light chain self-association; dimerization constants of the 17 proteins, calculated from a computer simulation of their behavior upon gel filtration, ranged from less than 10(3) to greater than 10(6) M-1. The **variable** region, more specifically the third hypervariable region, appears to be responsible for the variation in the dimerization constant. Association properties of light chains of known sequence suggest that the presence of an aromatic or **hydrophobic residue** at position 96 enhances dimer formation whereas a charged residue at that position results in light chains remaining stable monomers. The location of hypervariable residue 96 within the amino-terminal portion of the joining segment of the **variable** region suggests that the joining region may account for the variability of self-association of light chains and, moreover, that it has a function in determining the selective association of immunoglobulin polypeptide chains.

CT Amino Acid Sequence
***Binding Sites, Antibody**
Chromatography, Gel
Electrophoresis, Polyacrylamide Gel
Humans
***Immunoglobulin Variable Region**
*Immunoglobulins, Light-Chain
Immunoglobulins, Light-Chain: IM, immunology
*Immunoglobulins, kappa-Chain
Immunoglobulins, kappa-Chain: IM, immunology
Macromolecular Substances
Protein Binding
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.

CN 0 (**Binding** Sites, **Antibody**); 0 (Immunoglobulin **Variable** Region); 0 (Immunoglobulins, Light-Chain); 0 (Immunoglobulins, kappa-Chain); 0 (Macromolecular Substances)

ANSWER 6 OF 8 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1997:102459 CAPLUS
DN 126:130271
ED Entered STN: 13 Feb 1997
TI Production of single-chain Fv monomers and multimers
AU Filpula, David; McGuire, Jeffrey; Whitlow, Marc
CS Dep. Tumor Biol. Mol. Genet., Enzon Inc., Piscataway, NJ, 08854-3998, USA
SO Antibody Engineering (1996), 253-268. Editor(s): McCafferty, John; Hoogenboom, Hennie R.; Chiswell, Dave J. Publisher: IRL Press, Oxford, UK. CODEN: 63VUAO
DT Conference; General Review
LA English
CC 15-0 (Immunochemistry)
Section cross-reference(s): 16
AB A review with 32 refs. The authors describe their protocols for the design, construction, and purification of single-chain Fv proteins (scFv). They also describe the phenomenon of aggregation of scFv proteins to form multivalent Fv. Although the presence of scFv dimers and higher aggregates can be troublesome in the purification of scFv monomers, these stable Fv multimers have recently been identified as rearranged **multivalent antibody fragments** which may extend scFv technol. into bispecificity and crosslinking capabilities.
ST single chain Fv monomer multimer review
IT Immunoglobulins
RL: BPN (Biosynthetic preparation); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(single-chain Fv; production of single-chain Fv monomers and multimers

ANSWER 5 OF 8 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 3

AN 1996:581569 CAPLUS

DN 125:245193

ED Entered STN: 30 Sep 1996

TI **Multivalent antibody fragments** with high functional affinity for a tumor-associated carbohydrate antigen

AU Rheinnecker, Michael; Hardt, Christina; Ilag, Leodevico L.; Kufer, Peter; Gruber, Rudolf; Hoess, Adolf; Lupas, Andrei; Rottenberger, Christine; Plueckthun, Andreas; Pack, Peter

CS MorphoSys GmbH, Munich, Germany

SO Journal of Immunology (1996), 157(7), 2989-2997
CODEN: JOIMA3; ISSN: 0022-1767

PB American Association of Immunologists

DT Journal

LA English

CC 15-3 (Immunochemistry)

AB The authors report a human-derived self-assembling polypeptide based on the tetramerization domain of the human transcription factor p53, which can be fused to single-chain Fv Ab (scFv) fragments via a long and flexible hinge sequence of human origin, allowing exploitation of the functional affinity increase of binding to a ligand or cell surface with multimeric binding sites. This polypeptide was applied to the construction of a tetrameric scFv against the tumor-associated carbohydrate Ag Lewis Y (Fucα1→2Galβ1→4[Fucα1→3] GlcNAcβ1→3R). For comparison purposes, the corresponding scFv and dimeric mini-antibody, comprising the scFv fused via a flexible murine hinge to an artificial dimerization domain, were also created. The recombinant mini-antibody proteins were expressed in functional form in Escherichia coli and showed the expected m.w. of a dimer and tetramer, resp. Anal. of Lewis Y-binding behavior by surface plasmon resonance revealed specific but very weak binding of the scFv fragment. In contrast, both dimeric and tetrameric scFv fusion proteins exhibited an enormous gain in functional affinity that was greatest in the case of the tetrameric mini-antibody.

ST **multivalent antibody fragment** tumor carbohydrate antigen

IT Peptides, biological studies
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(self-assembling polypeptide in preparation of **multivalent antibody fragments** with affinity for tumor-associated carbohydrate antigen Lewis Y)

IT Antibodies
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(single-chain Fv fragments; self-assembling polypeptide in preparation of **multivalent antibody fragments** with affinity for tumor-associated carbohydrate antigen Lewis Y)

IT Blood-group substances
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Ley, self-assembling polypeptide in preparation of **multivalent antibody fragments** with affinity for tumor-associated carbohydrate antigen Lewis Y)

IT Proteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(fusion products, self-assembling polypeptide in preparation of **multivalent antibody fragments** with affinity for tumor-associated carbohydrate antigen Lewis Y)

IT Phosphoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(tumor suppressor, p53, peptide from tetramerization domain of;
self-assembling polypeptide in preparation of **multivalent antibody fragments** with affinity for tumor-associated carbohydrate antigen Lewis Y)

IT 182015-82-3DP, peptide conjugates
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(self-assembling polypeptide in preparation of **multivalent antibody fragments** with affinity for tumor-associated carbohydrate antigen Lewis Y)

ANSWER 9 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1993:558235 CAPLUS
DN 119:158235
ED Entered STN: 16 Oct 1993
TI **Multivalent** antigen-binding proteins
IN Whitlow, Marc D.; Wood, James F.; Hardman, Karl; Bird, Robert E.; Filpula, David; Rollence, Michele
PA Enzon, Inc., USA
SO PCT Int. Appl., 118 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07K015-28
ICS C07K003-20; C07H021-04; C12P021-08; C12N015-00; C12N015-03
CC 15-3 (Immunochemistry)
Section cross-reference(s): 3
FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9311161	A1	19930610	WO 1992-US9965	19921120
	W: AU, CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, SE				
	AU 9331789	A1	19930628	AU 1993-31789	19921120
	EP 617706	A1	19941005	EP 1993-900545	19921120
	EP 617706	B1	20011017		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, SE				
	JP 07501451	T2	19950216	JP 1992-510157	19921120
	EP 1136556	A1	20010926	EP 2001-109203	19921120
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, IE				
	AT 207080	E	20011115	AT 1993-900545	19921120
	ES 2165851	T3	20020401	ES 1993-900545	19921120
	US 6025165	A	20000215	US 1998-166750	19981005
	US 6027725	A	20000222	US 1998-166093	19981005
	US 6121424	A	20000919	US 1998-166094	19981005
	US 6103889	A	20000815	US 1998-172019	19981014
	US 6515110	B1	20030204	US 1999-443213	19991119
PRAI	US 1991-796936	A	19911125		
	EP 1993-900545	A3	19921120		
	US 1992-989846	B3	19921120		
	WO 1992-US9965	A	19921120		
	US 1995-392338	A3	19950222		
	US 1998-166094	A1	19981005		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 9311161	ICM	C07K015-28
	ICS	C07K003-20; C07H021-04; C12P021-08; C12N015-00; C12N015-03
EP 1136556	ECLA	C07K016/30; C07K016/44; C07K016/46D
US 6025165	ECLA	C07K001/22; C07K016/30; C07K016/44; C07K016/46D
US 6027725	ECLA	C07K001/22; C07K016/30; C07K016/44; C07K016/46D
US 6121424	ECLA	C07K001/22; C07K016/30; C07K016/44; C07K016/46D
US 6103889	ECLA	C07K016/30; C07K016/44; C07K016/46D

AB Compns. of, genetic constructions coding for, and methods for producing **multivalent** antigen-binding proteins are described and claimed. The antigen-binding proteins have ≥2 single-chain mols. comprising a) a 1st polypeptide comprising the binding portion of the variable region of an antibody heavy or **light chain**; b) a 2nd polypeptide comprising the variable region of an antibody chain and c) a peptide linker linking the 2 polypeptides into a single-chain mol. The methods include purification of compns. containing both monomeric and **multivalent** forms of single polypeptide chain mols., and production of **multivalent** proteins from purified monomers. Production of

multivalent proteins may occur by a concentration-dependent association of monomeric proteins, or by rearrangement of regions involving dissociation followed by association of different regions. Bivalent proteins, including homobivalent and heterobivalent proteins, are made in the present invention. Genetic sequences coding for the bivalent single-chain antigen-binding proteins are disclosed. Uses include all those appropriate for monoclonal and polyclonal antibodies and fragments thereof, including use as a bispecific antigen-binding mol.

ST **multivalent** antigen binding protein recombinant; **antibody fragment** fusion protein **multivalent**; bispecific antigen binding protein

IT Peptides, biological studies
RL: BIOL (Biological study)
(antibody variable regions linked by, in **multivalent** antigen-binding proteins)

IT Therapeutics
(associated with **multivalent** antigen-binding protein)

IT Deoxyribonucleic acid sequences
(for single-chain antigen-binding proteins)

IT Animal
(imaging of internal structure of, labeled **multivalent** antigen-binding protein for)

IT Chromatography, gel
(in separation of **multivalent** antigen-binding proteins from single-chain mols.)

IT Immunoassay
(**multivalent** antigen-binding proteins for)

IT Imaging
(of internal structure of animal, labeled **multivalent** antigen-binding protein for)

IT Protein sequences
(of single-chain antigen-binding proteins)

IT Antigens
RL: PREP (Preparation)
(proteins binding, **multivalent**, recombinant preparation and use of)

IT Antibodies
RL: BIOL (Biological study)
(variable regions of, linked by peptide linker, in **multivalent** antigen-binding proteins)

IT Proteins, specific or class
RL: BIOL (Biological study)
(SCA (single-chain antigen-binding), antibody variable regions linked by peptide linker as)

IT Antigens
RL: BIOL (Biological study)
(TAG-72 (tumor-associated glycoprotein 72), monoclonal antibody CC49 to, variable regions of, **multivalent** antigen-binding proteins containing)

IT Diagnosis
(agents, associated with **multivalent** antigen-binding protein)

IT Proteins, specific or class
RL: PREP (Preparation)
(antigen-binding, **multivalent**, recombinant preparation and use of)

IT Proteins, specific or class
RL: BIOL (Biological study)
(antigen-binding, labeled, **multivalent**, imaging of internal structure of animal with)

IT Chromatography, column and liquid
(cation-exchange, in separation of **multivalent** antigen-binding proteins from single-chain mols.)

IT Gene, animal
RL: PREP (Preparation)

(chimeric, for single-chain antigen-binding protein, in **multivalent** antigen-binding protein preparation)

IT Immunoassay
(competitive, **multivalent** antigen-binding proteins for)

IT Therapeutics
(immuno-, **multivalent** antigen-binding proteins for)

IT Purification
(immunoaffinity, **multivalent** antigen-binding proteins for)

IT Antibodies
RL: BIOL (Biological study)
(monoclonal, variable regions of, linked by peptide linker, in **multivalent** antigen-binding proteins)

IT 150067-45-1
RL: PRP (Properties)
(amino acid sequence of)

IT 150067-49-5
RL: PRP (Properties)
(amino acid sequence of and expression of DNA for, in Escherichia coli)

IT 150067-51-9 150067-53-1
RL: PRP (Properties)
(amino acid sequence of and heterodimer Fv antibody construct preparation from)

IT 150067-41-7 150067-43-9
RL: PRP (Properties)
(amino acid sequence of and mixed-fragment bivalent antigen-binding protein preparation with)

IT 131641-67-3, PolyCAT A
RL: PRP (Properties)
(antigen-binding proteins purification on)

IT 130838-28-7 150243-58-6 150243-59-7 150243-60-0
RL: PRP (Properties)
(as linker peptide in **multivalent** antigen-binding proteins)

IT 50-01-1, Guanidine hydrochloride 57-13-6, Urea, biological studies
RL: PRP (Properties)
(ethanol and, as dissociating agents in preparation of **multivalent** antigen-binding proteins)

IT 64-17-5, Ethanol, biological studies
RL: PRP (Properties)
(guanidine hydrochloride and, as dissociating agents in preparation of **multivalent** antigen-binding proteins)

IT 2321-07-5, Fluorescein
RL: PRP (Properties)
(monoclonal antibody 4-4-20 to, variable regions of, **multivalent** antigen-binding proteins containing)

IT 150067-48-4
RL: PROC (Process)
(nucleotide sequence and expression of, in Escherichia coli)

IT 150067-44-0
RL: PRP (Properties)
(nucleotide sequence of)

IT 150067-46-2
RL: PRP (Properties)
(nucleotide sequence of and antigen-binding dimer preparation in relation to)

IT 150067-50-8 150067-52-0
RL: PRP (Properties)
(nucleotide sequence of and heterodimer Fv antibody construct preparation with)

IT 150067-40-6 150067-42-8
RL: PRP (Properties)
(nucleotide sequence of and mixed-fragment bivalent antigen-binding protein preparation with)

IT 150067-47-3DP, crosslinked

RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)

=>

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? begin 5,73,155,399
17mar05 13:01:11 User208760 Session D2584.2
$0.00 0.102 DialUnits File410
$0.00 Estimated cost File410
$0.03 TELNET
$0.03 Estimated cost this search
$0.41 Estimated total session cost 0.211 DialUnits

09/368,989
Search by Phil G.
L/Cook 3/21/05

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? s janussin?		
S1	3	JANUSSIN?
? rd s1		
...completed examining records		
S2	2	RD S1 (unique items)

? t s2/7/all

2/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0006051507 BIOSIS NO.: 198885020398
ALKALOIDS OF STRYCHNOS-JOHNSONII
AUTHOR: MASSIOT G (Reprint); THEPENIER P; JACQUIER M-J; LE MEN-OLIVIER L; VERPOORTE R; DELAUDE C
AUTHOR ADDRESS: FAC PHARM, UA CNRS NO 492, 51 RUE COGNACQ-JAY, 51096 REIMS CEDEX, FRANCE**FRANCE
JOURNAL: Phytochemistry (Oxford) 26 (10): p2839-2846 1987
ISSN: 0031-9422
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: Twenty-three alkaloids have been identified in the root bark and stem bark of Strychnos johnsonii from Zaire. They are demethoxycarbonyl-3,14-dihydro-gambirtannine, anguistine, dihydro-cycloakagerine. O-ethylakagerine, O-ethylakagerine lactone, dihydro decussine, normalindine, oxojanussine, tetrahydroalstonial, ajmalicinial, norepimalindine, norharman, harman, akagerine, akagerine lactone, **janussines** A and B, tetrahydro akagerine, demethoxycarbonyl-3,14,15,16,17,18-hexahydro-gambirtannine, dihydrocorynantheol, anthirine lactone, anthirine and isoanthirine.

2/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0004539404 BIOSIS NO.: 198529068303
JANUSSINES A AND B FROM STRYCHNOS-JOHNSONII A CROSS POINT IN THE BIOGENESIS OF QUASI-DIMERIC ALKALOIDS
AUTHOR: MASSIOT G (Reprint); THEPENIER P; JACQUIER M J; DELAUDE C; LE MEN-OLIVIER L; VERPOORTE R
AUTHOR ADDRESS: UA AU CNRS NO 492-CESNAB-UNIVERSITE DE REIMS, FACULTE DE

PHARMACIE, F51096 REIMS CEDEX, FRANCE**FRANCE
JOURNAL: Tetrahedron Letters 26 (20): p2441-2444 1985
ISSN: 0040-4039
DOCUMENT TYPE: Article
RECORD TYPE: Citation
LANGUAGE: ENGLISH
? s janusin?
S3 136 JANUSIN?
? rd s3
...examined 50 records (50)
...examined 50 records (100)
...completed examining records
S4 108 RD S3 (unique items)
? s s4 and antibod?
108 S4
1978760 ANTIBOD?
S5 36 S4 AND ANTIBOD?
? rd s5
...completed examining records
S6 36 RD S5 (unique items)
? t s6/7/all

6/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012646392 BIOSIS NO.: 200000364705
The two faces of perineuronal nets
AUTHOR: Viggiano Davide (Reprint)
AUTHOR ADDRESS: School of Medicine, Institute of Human Anatomy, Second University of Naples, Via L. Armanni 5, 80138, Naples, Italy**Italy
JOURNAL: Neuroreport 11 (10): p2087-2090 14 July, 2000 2000
MEDIUM: print
ISSN: 0959-4965
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Perineuronal nets are extracellular structures enwrapping the soma and proximal dendrites of some neurons known to be parvalbumin immunoreactive. The composition of the nets is not completely known, but it can change between different neurons. We studied the heterogeneous composition of a specific component of perineuronal nets, the signaling molecule **Janusin** (or Tenascin R), by means of a double immunofluorescence using lectin from Wisteria floribunda as a general marker for perineuronal nets and an ***antibody*** against ***Janusin*** . The presence of two kinds of perineuronal nets, one rich in **Janusin** (the majority) and a second one devoid of this glycoprotein, indicates differential roles of these neurons, as well as differences in their afferents, or a difference in their functional state.

6/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008902749 BIOSIS NO.: 199396067165
Influence of **janusin** and tenascin on growth cone behavior in vitro
AUTHOR: Taylor J (Reprint); Pesheva P; Schachner M
AUTHOR ADDRESS: Dep. Neurobiol., Swiss Federal Inst. Technol., Honggerberg, 8093 Zurich, Switzerland**Switzerland
JOURNAL: Journal of Neuroscience Research 35 (4): p347-362 1993
ISSN: 0360-4012
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Janusin** and tenascin are glia-derived, structurally related, extracellular matrix glycoproteins of the J1 family that are expressed in vivo at times and in locations where active neurite outgrowth occurs, but also when the formation or stabilization of cytoarchitectonic boundaries appears to be in operation. To resolve this apparent functional dichotomy, we have studied the behavioral response of growth cones, growing in culture on the permissive substrate laminin, to ***janusin*** and tenascin, by video time lapse microscopy. When **janusin** and tenascin were offered as sharp substrate boundaries, dorsal root ganglion (DRG) and retinal ganglion neuron growth cones avoided growing on these molecules, but were not induced to collapse. On the other hand, when **janusin** and tenascin were offered, in a mixture with laminin, as uniform substrates, DRG growth cones displayed a collapsed morphology and were able to advance at a faster rate than on laminin alone. In contrast, the outgrowth of retinal ganglion neuron growth cones was completely inhibited under these conditions, underscoring a cell type specificity in the response of growth cones to these molecules. Using several monoclonal ***antibodies*** binding to distinct epitopes on the tenascin molecule, we have identified two domains responsible for growth cone repulsion, on epidermal growth factor (EGF)-like repeats 3-5 and fibronectin type III homologous repeats 4 and 5. These domains are different from the one previously recognized to be involved in neurite outgrowth on a uniform tenascin substrate. We conclude that both molecules may promote or retard growth cone advance, depending on the spatial expression pattern and the neuronal cell type.

6/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008843298 BIOSIS NO.: 199396007714
Localization of **Janusin** mRNA in the central nervous system of the developing and adult mouse
AUTHOR: Wintergerst Eva Sabine (Reprint); Fuss Babette; Bartsch Udo
AUTHOR ADDRESS: Dep. Neurobiology, Swiss Federal Inst. Technology, Honggerberg, 8093 Zurich, Switzerland**Switzerland
JOURNAL: European Journal of Neuroscience 5 (4): p299-310 1993
ISSN: 0953-816X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Janusin** (formerly termed J1-160/180) is an oligodendrocyte-derived extracellular matrix molecule which is restricted to the central nervous system and which is expressed late during development (Pesheva et al., J. Cell Biol., 1765-1778, 1989). To gain insights into the molecule's morphogenetic functions and to identify its cellular source in vivo, we have studied the localization of **janusin** messenger RNA in the optic nerve, retina and spinal cord and the expression of **janusin** protein in the spinal cord of developing and adult mice. Moreover, we have analyzed optic nerve cell cultures and retinal cell suspensions in double-labelling experiments using a **janusin**-specific anti-sense complementary RNA probe and cell type-specific **antibodies** to identify the cell types containing ***janusin*** transcripts. In developing animals, oligodendrocytes were strongly labelled with the **janusin** anti-sense cRNA probe during the period of myelination. The number of labelled cells and intensity of the hybridization signal decreased significantly with increasing age. Interestingly, expression of **janusin** was not confined to oligodendrocytes. Some neuronal cell types and type-2 astrocytes present in optic nerve cell cultures also contained ***janusin*** transcripts. In contrast to oligodendrocytes, the number and labelling intensity of neurons containing **janusin** transcripts remained constant during postnatal development and into adulthood. Expression of ***janusin*** protein in the spinal cord was developmentally regulated, with a peak of expression in 2- or 3-week-old animals. The molecule was visible in the

white and grey matter. In myelinated regions it was associated with myelinated fibres and accumulated at nodes of Ranvier. These observations suggest that **janusin** may be of functional relevance for myelination.

6/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008201724 BIOSIS NO.: 199293044615
BISPECIFIC SINGLE CHAIN MOLECULES **JANUSINS** TARGET CYTOTOXIC LYMPHOCYTES ON HIV INFECTED CELLS
AUTHOR: TRAUNECKER A (Reprint); LANZAVECCHIA A; KARJALAINEN K
AUTHOR ADDRESS: BASEL INST IMMUNOL, CH-4005 BASEL, SWITZ**SWITZERLAND
JOURNAL: EMBO (European Molecular Biology Organization) Journal 10 (12): p 3655-3660 1991
ISSN: 0261-4189
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: The human immunodeficiency virus type 1 (HIV-1) uses cell surface CD4 as a receptor to infect susceptible cells. Therefore, different forms of soluble CD4 (sCD4) molecules have been developed recently for potential therapeutic purposes. Here we describe a novel design of sCD4 molecules which exploit cytotoxic T cells as their effector function. The principle of bispecific **antibodies** was exploited and further developed to create new bispecific reagents which could retarget cytotoxic T cells of any specificity and thus, induce killing of HIV-1 infected cells. The most advanced molecules, ***Janusins*** , contain in one polypeptide chain the first two N-terminal CD4 domains and single chain combining site against the human CD3 complex (FvCD3).

6/7/5 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

05619241 EMBASE No: 1994023157
Perineuronal nets - A specialized form of extracellular matrix in the adult nervous system
Celio M.R.; Blumcke I.
Inst. Histology/General Embryology, University of Fribourg, Rte. Albert Gockel,CH-1075 Fribourg Switzerland
Brain Research Reviews (BRAIN RES. REV.) (Netherlands) 1994, 19/1 (128-145)
CODEN: BRERD ISSN: 0165-0173
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

One century ago, Camillo Golgi described 'perineuronal nets' enwrapping the cell bodies and proximal dendrites of certain neurons in the adult mammalian central nervous system and suggested that they represent a supportive and protective scaffolding. Although other neuroanatomists validated the existence of these nets on selected neurons in the adult brain, there was a lack of agreement on their origins, composition and function. The application of modern molecular and ultrastructural methods has brought new insights and a renewed interest in these classic observations. Recent data suggest that perineuronal nets result from the visualization of extracellular matrix molecules that are confined to the space interposed between glial processes and the nerve cells that they outline. The material confined to these spaces can be visualized selectively by ***antibodies*** directed to glycoproteins (e.g., tenascin and restriction/ ***janusin***), proteoglycans (e.g., chondroitin sulfates), markers for hyaluronan as well as by lectins recognizing N-acetylgalactosamine and by monoclonal **antibodies** directed to

epitopes on unknown molecules (e.g., HNK-1, VC1.1 and Cat 301). This review examines the emerging clarification of classical observations of perineuronal nets and the functional implications suggested by their molecular composition. Also discussed are studies that further extend observations on the time of development and of the specificity in the occurrence of perineuronal nets. In the adult brain the molecules constituing the 'perineuronal nets of matrix' cold serve as recognition molecules between certain neurons and their surrounding cells and participate in the selection and consolidation of their relationships.

6/7/6 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14954948 PMID: 12954529
Region and lamina-specific distribution of extracellular matrix proteoglycans, hyaluronan and tenascin-R in the mouse hippocampal formation.
Bruckner Gert; Grosche Jens; Hartlage-Rubsamen Maike; Schmidt Sandra; Schachner Melitta
Department of Neurochemistry, Paul Flechsig Institute for Brain Research, University of Leipzig, Jahnallee 59, D-04109 Leipzig, Germany. brug@server3.medizin.uni-leipzig.de
Journal of chemical neuroanatomy (Netherlands) Aug 2003, 26 (1) p37-50, ISSN 0891-0618 Journal Code: 8902615
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The extracellular matrix is known to show region-specific characteristics in the adult brain. Our comparative cytochemical study is focused on the laminar organisation of major extracellular matrix constituents in the murine hippocampal formation, including the regions CA1, CA2 and CA3 of the hippocampus proper, the dentate gyrus, the subiculum and the presubiculum. Components related to chondroitin sulphate proteoglycans were detected by N-acetylgalactosamine-binding Wisteria floribunda agglutinin, colloidal iron staining, and **antibodies** to different proteoglycan domains, including the Cat-301 and Cat-315 epitopes of aggrecan, as well as neurocan, brevican and phosphacan. The distribution patterns of these components were correlated with the patterns revealed for hyaluronan and the brain-specific extracellular matrix glycoprotein, tenascin-R, known to be ligands of extracellular matrix proteoglycans. Lectin binding clearly labelled perineuronal nets of the extracellular matrix around interneurons, which were preferentially located within or near the principal cell layers in all regions. In the hippocampus proper, the CA2 subfield showed an intense labelling of the neuropil around pyramidal cell bodies and the neuropil zones in the strata oriens and radiatum. These patterns were also seen after immunoreaction for chondroitin proteoglycan domains, brevican and phosphacan, as well as after detection of hyaluronan and tenascin-R. Characteristic laminar and intralaminar patterns were additionally expressed in the neuropil in all regions. In the dentate gyrus, the staining intensity for brevican, phosphacan and tenascin-R was predominant in the middle molecular layer, and for Cat-315 in the inner molecular layer, whereas immunoreactivity for neurocan increased within the outer molecular layer towards the hippocampal fissure. Our findings indicate that proteoglycans, hyaluronan and tenascin-R show differential patterns of co-expression in the individual regions and laminae of the hippocampal formation. The inhomogeneous composition of these major components suggests that the extracellular matrix is specifically adapted to the functional domains of intrahippocampal connections and afferent fibre systems.
Record Date Created: 20030904
Record Date Completed: 20031106

6/7/7 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
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14546621 PMID: 12504595
Tenascin-R induces actin-rich microprocesses and branches along neurite shafts.
Zacharias Ute; Leuschner Roland; Norenberg Ursel; Rathjen Fritz G
Max-Delbruck-Centrum fur Molekulare Medizin, Robert-Rossle-Strasse 10, D-13092 Berlin, Germany. uzachar@mdc-berlin.de
Molecular and cellular neurosciences (United States) Dec 2002, 21 (4) p626-33, ISSN 1044-7431 Journal Code: 9100095
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The formation of protrusions along the shaft of neurites might be important in the establishment and refinement of neuronal connections during development. In a search for extracellular signals that affect the formation of microprocesses along neurites we found that the ECM glycoprotein tenascin-R (TN-R) but not other ECM glycoproteins increased the percentage of tectal neurons with actin-rich microprocesses and side branches. Longer actin-based microprocesses were also invaded by microtubuli in their proximal part. The formation of microprocesses by TN-R extending laterally along the neuritic shaft was time- and dose-dependent. In addition to the induction of microprocesses, TN-R increased the size of the growth cone of tectal neurons. A cross-species experiment in combination with blocking **antibodies** demonstrated that the TN-R-induced effects are mediated by the Ig superfamily member contactin. These observations suggest that TN-R via its neuronal receptor contactin might induce a transition from long-distance growth of tectal interneurons to differentiation, including the formation of microprocesses.
Record Date Created: 20021230
Record Date Completed: 20030321

6/7/8 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2005 The Dialog Corp. All rts. reserv.

14099041 PMID: 11875281
Immunocytological localization of the HNK-1 carbohydrate in murine cerebellum, hippocampus and spinal cord using monoclonal **antibodies** with different epitope specificities.
Rollenhagen A; Czaniera R; Albert M; Wintergerst E S; Schachner M
Zentrum fur Molekulare Neurobiologie, Universitat Hamburg, Martinistr. 52, D20246 Hamburg, Germany.
Journal of neurocytology (United States) Apr 2001, 30 (4) p337-51, ISSN 0300-4864 Journal Code: 0364620
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The HNK-1 carbohydrate, an unusual 3'-sulfated glucuronic acid epitope characteristic of many neural recognition molecules, serves as a ligand in neural cell interactions and is differentially expressed in the quadriceps and saphenous branches of the femoral nerve in the PNS of adult mice. Based on these observations, we investigated the possibility that the HNK-1 carbohydrate may be differentially distributed in neurons and fiber tracts also in the CNS thereby contributing to different targeting and guidance mechanisms. We have used ***antibodies*** with different HNK-1 epitope specificities to probe for subtle differences in expression patterns. In the adult mouse cerebellum the HNK-1 carbohydrate is detectable in stripe-like compartments in the molecular and Purkinje cell layers, whereas N-CAM and its associated alpha2,8 polysialic acid does not show this compartmentation. In the adult hippocampus, the HNK-1 carbohydrate

localizes to perineuronal nets of inhibitory interneurons and marks the inner third of the molecular layer of the dentate gyrus. In the adult spinal cord, HNK-1 labeling is most pronounced in gray matter areas. White matter enriched regions show differential labeling with regard to fiber tracts and ***antibody*** specificity. Whereas the different **antibodies** do not show differences in staining in the cerebellum and the hippocampus, they show differences in staining pattern of fiber tracts and motoneurons in the spinal cord. The HNK-1 expression pattern also differed in the adult spinal cord from that observed at embryonic day 14 and postnatal day 14. Our observations suggest a functional role in the specification of functionally discrete compartments in different areas of the CNS and during development.
Record Date Created: 20020304
Record Date Completed: 20020523

6/7/9 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13909715 PMID: 11598921
Immunoelectron microscopic localization of the neural recognition molecules L1, NCAM, and its isoform NCAM180, the NCAM-associated polysialic acid, beta1 integrin and the extracellular matrix molecule tenascin-R in synapses of the adult rat hippocampus.
Schuster T; Krug M; Stalder M; Hackel N; Gerardy-Schahn R; Schachner M
Zentrum fur Molekulare Neurobiologie, Universitat Hamburg, Hamburg, Germany. Thomas.schuster@zmnh.uni-hamburg.de
Journal of neurobiology (United States) Nov 5 2001, 49 (2) p142-58, ISSN 0022-3034 Journal Code: 0213640
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
We have investigated the possibility that morphologically different excitatory glutamatergic synapses of the "trisynaptic circuit" in the adult rodent hippocampus, which display different types of long-term potentiation (LTP), may express the immunoglobulin superfamily recognition molecules L1 and NCAM, the extracellular matrix molecule tenascin-R, and the extracellular matrix receptor constituent beta1 integrin in a differential manner. The neural cell adhesion molecules L1, NCAM (all three major isoforms), NCAM180 (the largest major isoform with the longest cytoplasmic domain), beta1 integrin, polysialic acid (PSA) associated with NCAM, and tenascin-R were localized by pre-embedding immunostaining procedures in the CA3/CA4 region (mossy fiber synapses) and in the dentate gyrus (spine synapses) of the adult rat hippocampus. Synaptic membranes of mossy fiber synapses where LTP is expressed presynaptically did not show detectable levels of immunoreactivity for any of the molecules/epitopes studied. L1, NCAM, and PSA, but not NCAM180 or beta1 integrin, were detectable on axonal membranes of fasciculating mossy fibers. In contrast to mossy fiber synapses, spine synapses in the outer third of the molecular layer of the dentate gyrus, which display postsynaptic expression mechanisms of LTP, were both immunopositive and immunonegative for NCAM, NCAM180, beta1 integrin, and PSA. Those spine synapses postsynaptically immunoreactive for NCAM or PSA also showed immunoreactivity on their presynaptic membranes. NCAM180 was not detectable presynaptically in spine synapses. L1 could not be found in spine synapses either pre- or postsynaptically. Also, the extracellular matrix molecule tenascin-R was not detectable in synaptic clefts of all synapses tested, but was amply present between fasciculating axons, axon-astrocyte contact areas, and astrocytic gap junctions. Differences in expression of the membrane-bound adhesion molecules at both types of synapses may reflect the different mechanisms for induction and/or maintenance of synaptic plasticity. Copyright 2001 John Wiley & Sons, Inc.
Record Date Created: 20011012
Record Date Completed: 20011213

6/7/10 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13720674 PMID: 11069908

CALEB binds via its acidic stretch to the fibrinogen-like domain of tenascin-C or tenascin-R and its expression is dynamically regulated after optic nerve lesion.

Schumacher S; Jung M; Norenberg U; Dorner A; Chiquet-Ehrismann R; Stuermer C A; Rathjen F G

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Journal of biological chemistry (United States) Mar 9 2001, 276 (10) p7337-45, ISSN 0021-9258 Journal Code: 2985121R

Publishing Model Print-Electronic

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Recently, we described a novel chick neural transmembrane glycoprotein, which interacts with the extracellular matrix proteins tenascin-C and tenascin-R. This protein, termed CALEB, contains an epidermal growth factor-like domain and appears to be a novel member of the epidermal growth factor family of growth and differentiation factors. Here we analyze the interaction between CALEB and tenascin-C as well as tenascin-R in more detail, and we demonstrate that the central acidic peptide segment of CALEB is necessary to mediate this binding. The fibrinogen-like globe within tenascin-C or -R enables both proteins to bind to CALEB. We show that two isoforms of CALEB in chick and rodents exist that differed in their cytoplasmic segments. To begin to understand the in vivo function of CALEB and since in vitro **antibody** perturbation experiments indicated that CALEB might be important for neurite formation, we analyzed the expression pattern of the rat homolog of CALEB during development of retinal ganglion cells, after optic nerve lesion and during graft-assisted retinal ganglion cell axon regeneration by in situ hybridization. These investigations demonstrate that CALEB mRNA is dynamically regulated after optic nerve lesion and that this mRNA is expressed in most developing and in one-third of the few regenerating (GAP-43 expressing) retinal ganglion cells.

Record Date Created: 20010525

Record Date Completed: 20010719

Date of Electronic Publication: 20001107

6/7/11 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13480740 PMID: 10446214

Functional interactions of the immunoglobulin superfamily member F11 are differentially regulated by the extracellular matrix proteins tenascin-R and tenascin-C.

Zacharias U; Norenberg U; Rathjen F G

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Journal of biological chemistry (UNITED STATES) Aug 20 1999, 274 (34) p24357-65, ISSN 0021-9258 Journal Code: 2985121R

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

The axon-associated protein F11 is a GPI-anchored member of the immunoglobulin superfamily that promotes axon outgrowth and that shows a complex binding pattern toward multiple cell surface and extracellular matrix proteins including tenascin-R and tenascin-C. In this study, we demonstrate that tenascin-R and tenascin-C differentially modulate cell

adhesion and neurite outgrowth of tectal cells on F11. While soluble tenascin-R increases the number of attached cells and the percentage of cells with neurites on immobilized F11, tenascin-C stimulates cell attachment to a similar extent but decreases neurite outgrowth. The cellular receptor interacting with F11 has been previously identified as NrCAM; however, in the presence of tenascin-R or tenascin-C cell attachment and neurite extension are independent of NrCAM. ***Antibody*** perturbation experiments indicate that beta(1) integrins instead of NrCAM function as receptor for neurite outgrowth of tectal cells on an F11.TN-R complex. Cellular binding assays support the possibility that the interaction of F11 to NrCAM is blocked in the presence of tenascin-R and tenascin-C. Furthermore, a sandwich binding assay demonstrates that tenascin-R and tenascin-C are able to form larger molecular complexes and to link F11 polypeptides by forming a molecular bridge. These results suggest that the molecular interactions of F11 might be regulated by the presence of tenascin-R and tenascin-C.

Record Date Created: 19990909

Record Date Completed: 19990909

6/7/12 (Item 7 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

13423634 PMID: 10383637

Tenascin-R interferes with integrin-dependent oligodendrocyte precursor cell adhesion by a ganglioside-mediated signalling mechanism.

Probstmeier R; Michels M; Franz T; Chan B M; Pesheva P

Department of Biochemistry, Institute of Animal Anatomy and Physiology, University of Bonn, 53115, Germany.

European journal of neuroscience (FRANCE) Jul 1999, 11 (7) p2474-88, ISSN 0953-816X Journal Code: 8918110

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Oligodendrocyte (OL) lineage progression is characterized by the transient expression of the disialoganglioside GD3 by OL precursor (preOL) cells followed by the sequential expression of myelin-specific lipids and proteins. Whereas GD3+ preOLs are highly motile cells, the migratory capacity of OLs committed to terminal differentiation is strongly reduced, and we have recently shown that the extracellular matrix protein tenascin-R (TN-R) promotes the stable adhesion and differentiation of O4+ OLs by a sulphatide-mediated autocrine mechanism (O4 is a monoclonal **antibody** recognizing sulphatides/seminolipids expressed by OLs and in myelin). Using culture conditions that allow the isolation of mouse OLs at distinct lineage stages, here we demonstrate that TN-R is antiadhesive for GD3+ preOLs and inhibits their integrin-dependent adhesion to fibronectin (FN) by a disialoganglioside-mediated signalling mechanism affecting the tyrosine phosphorylation of the focal adhesion kinase. This responsive mechanism appears to be common to various cell types expressing disialogangliosides as: (i) disialogangliosides interfered with the inhibition of cell adhesion of different neural and non-neural cells on substrata containing TN-R and FN or RGD-containing FN fragments. TN-R interacted specifically with disialoganglioside-expressing cells or immobilized gangliosides, and ganglioside treatment of TN-R substrata resulted in a delayed preOL cell detachment as a function of time. We conclude that OL response to one and the same signal in the extracellular matrix critically depends on the molecular repertoire expressed by OLs at different lineage stages and could thus define their final positioning.

Record Date Created: 19990730

Record Date Completed: 19990730

6/7/13 (Item 8 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

13387034 PMID: 10341229

Mice deficient for tenascin-R display alterations of the extracellular matrix and decreased axonal conduction velocities in the CNS.

Weber P; Bartsch U; Rasband M N; Czaniera R; Lang Y; Bluethmann H; Margolis R U; Levinson S R; Shrager P; Montag D; Schachner M

Department of Neurobiology, Swiss Federal Institute of Technology, Honggerberg, CH 8093 Zurich, Switzerland.

Journal of neuroscience - the official journal of the Society for Neuroscience (UNITED STATES) Jun 1 1999, 19 (11) p4245-62, ISSN 1529-2401 Journal Code: 8102140

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Tenascin-R (TN-R), an extracellular matrix glycoprotein of the CNS, localizes to nodes of Ranvier and perineuronal nets and interacts in vitro with other extracellular matrix components and recognition molecules of the immunoglobulin superfamily. To characterize the functional roles of TN-R in vivo, we have generated mice deficient for TN-R by homologous recombination using embryonic stem cells. TN-R-deficient mice are viable and fertile. The anatomy of all major brain areas and the formation and structure of myelin appear normal. However, immunostaining for the chondroitin sulfate proteoglycan phosphacan, a high-affinity ligand for TN-R, is weak and diffuse in the mutant when compared with wild-type mice. Compound action potential recordings from optic nerves of mutant mice show a significant decrease in conduction velocity as compared with controls. However, at nodes of Ranvier there is no apparent change in expression and distribution of Na+ channels, which are thought to bind to TN-R via their beta2 subunit. The distribution of carbohydrate epitopes of perineuronal nets recognized by the lectin Wisteria floribunda or **antibodies** to the HNK-1 carbohydrate on somata and dendrites of cortical and hippocampal interneurons is abnormal. These observations indicate an essential role for TN-R in the formation of perineuronal nets and in normal conduction velocity of optic nerve.

Record Date Created: 19990616

Record Date Completed: 19990616

6/7/14 (Item 9 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

13259321 PMID: 9880601

Tenascin-R inhibits the growth of optic fibers in vitro but is rapidly eliminated during nerve regeneration in the salamander Pleurodeles waltl.

Becker C G; Becker T; Meyer R L; Schachner M

Zentrum fur Molekulare Neurobiologie Hamburg, Universitat Hamburg, D-20246 Hamburg, Germany.

Journal of neuroscience - the official journal of the Society for Neuroscience (UNITED STATES) Jan 15 1999, 19 (2) p813-27, ISSN 0270-6474 Journal Code: 8102140

Contract/Grant Number: NS26750; NS; NINDS

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Tenascin-R is a multidomain molecule of the extracellular matrix in the CNS with neurite outgrowth inhibitory functions. Despite the fact that in amphibians spontaneous axonal regeneration of the optic nerve occurs, we show here that the molecule appears concomitantly with myelination during metamorphosis and is present in the adult optic nerve of the salamander Pleurodeles waltl by immunoblots and immunohistochemistry. In vitro, adult retinal ganglion cell axons were not able to grow from retinal explants on

a tenascin-R substrate or to cross a sharp substrate border of tenascin-R in the presence of laminin, indicating that tenascin-R inhibits regrowth of retinal ganglion cell axons. After an optic nerve crush, immunoreactivity for tenascin-R was reduced to undetectable levels within 8 d. Immunoreactivity for the myelin-associated glycoprotein (MAG) was also diminished by that time. Myelin was removed by phagocytosing cells at 8-14 d after the lesion, as demonstrated by electron microscopy. Tenascin-R immunoreactivity was again detectable at 6 months after the lesion, correlated with remyelination as indicated by MAG immunohistochemistry. Regenerating axons began to repopulate the distal lesioned nerve at 9 d after a crush and grew in close contact with putative astrocytic processes in the periphery of the nerve, close to the pia, as demonstrated by anterograde tracing. Thus, the onset of axonal regrowth over the lesion site was correlated with the removal of inhibitory molecules in the optic nerve, which may be necessary for successful axonal regeneration in the CNS of amphibians.

Record Date Created: 19990216
Record Date Completed: 19990216

6/7/15 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13103665 PMID: 11077416

Postnatal development of perineuronal nets in wild-type mice and in a mutant deficient in tenascin-R.

Bruckner G; Grosche J; Schmidt S; Hartig W; Margolis R U; Delpech B; Seidenbecher C I; Czaniera R; Schachner M

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Journal of comparative neurology (UNITED STATES) Dec 25 2000, 428 (4) p616-29, ISSN 0021-9967 Journal Code: 0406041

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

The extracellular matrix glycoprotein tenascin-R (TN-R), colocalizing with hyaluronan, phosphacan, and aggregating chondroitin sulphate proteoglycans in the white and grey matter, is accumulated in perineuronal nets that surround different types of neurons in many brain regions. To characterize the role of TN-R in the formation of perineuronal nets, we studied their postnatal development in wild-type mice and in a TN-R knock-out mutant by using the lectin Wisteria floribunda agglutinin and an **antibody** to nonspecified chondroitin sulphate proteoglycans as established cytochemical markers. We detected the matrix components TN-R, hyaluronan, phosphacan, neurocan, and brevican in the perineuronal nets of cortical and subcortical regions. In wild-type mice, lectin-stained, immature perineuronal nets were first seen on postnatal day 4 in the brainstem and on day 14 in the cerebral cortex. The staining intensity of these nets for TN-R, hyaluronan, phosphacan, neurocan, and brevican was extremely weak or not distinguishable from that of the surrounding neuropil. However, all markers showed an increase in staining intensity of perineuronal nets reaching maximal levels between postnatal days 21 and 40. In TN-R-deficient animals, the perineuronal nets tended to show a granular component within their lattice-like structure at early stages of development. Additionally, the staining intensity in perineuronal nets was reduced for brevican, extremely low for hyaluronan and neurocan, and virtually no immunoreactivity was detectable for phosphacan. The granular configuration of perineuronal nets became more predominant with advancing age of the mutant animals, indicating the continued abnormal aggregation of chondroitin sulphate proteoglycans complexed with hyaluronan. As shown by electron microscopy in the cerebral cortex, the disruption of perineuronal nets was not accompanied by apparent changes in the synaptic structure on net-bearing neurons. The regional distribution patterns and the temporal course of development of perineuronal nets were not obviously changed in

the mutant. We conclude that the lack of TN-R initially and continuously disturbs the molecular scaffolding of extracellular matrix components in perineuronal nets. This may interfere with the development of the specific micromilieu of the ensheathed neurons and adjacent glial cells and may also permanently change their functional properties. Copyright 2000 Wiley-Liss, Inc.
Record Date Created: 20001208
Record Date Completed: 20010104

6/7/16 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13035120 PMID: 10998116
The extracellular matrix molecule tenascin-R and its HNK-1 carbohydrate modulate perisomatic inhibition and long-term potentiation in the CA1 region of the hippocampus.
Saghatelyan A K; Gorissen S; Albert M; Hertlein B; Schachner M; Dityatev A
Zentrum fur Molekulare Neurobiologie, Universitat Hamburg, Martinistrasse 52, D-20246 Hamburg, Germany.
European journal of neuroscience (FRANCE) Sep 2000, 12 (9) p3331-42, ISSN 0953-816X Journal Code: 8918110
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Perisomatic inhibition of pyramidal cells regulates efferent signalling from the hippocampus. The striking presence of HNK-1, a carbohydrate expressed by neural adhesion molecules, on perisomatic interneurons and around somata of CA1 pyramidal neurons led us to apply monoclonal HNK-1 ***antibodies*** to acute murine hippocampal slices. Injection of these **antibodies** decreased GABAA receptor-mediated perisomatic inhibitory postsynaptic currents (pIPSCs) but did not affect dendritic IPSCs or excitatory postsynaptic currents. The decrease in the mean amplitude of evoked pIPSCs by HNK-1 **antibodies** was accompanied by an increase in the coefficient of variation of pIPSC amplitude, number of failures and changes in frequency but not amplitude of miniature IPSCs, suggesting that HNK-1 ***antibodies*** reduced efficacy of evoked GABA release. HNK-1 **antibodies** did not affect pIPSCs in knock-out mice deficient in the extracellular matrix molecule tenascin-R which carries the HNK-1 carbohydrate as analysed by immunoblotting in synaptosomal fractions prepared from the CA1 region of the hippocampus. For control, HNK-1 **antibody** was applied to acute sections of mice deficient in the neural cell adhesion molecule NCAM, another potential carrier of HNK-1, and resulted in decrease of pIPSCs as observed in wild-type mice. Reduction in perisomatic inhibition is expected to promote induction of long-term potentiation (LTP) by increasing the level of depolarization during theta-burst stimulation. Indeed, LTP was increased by HNK-1 ***antibody*** applied before stimulation. Moreover, LTP was reduced by an HNK-1 peptide mimic, but not control peptide. These results provide first evidence that tenascin-R and its associated HNK-1 carbohydrate modulate perisomatic inhibition and synaptic plasticity in the hippocampus.
Record Date Created: 20001020
Record Date Completed: 20001107

6/7/17 (Item 12 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12853919 PMID: 10793198
Morphology of perineuronal nets in tenascin-R and parvalbumin single and double knockout mice.
Haunso A; Ibrahim M; Bartsch U; Letiembre M; Celio M R; Menoud P

Institute of Histology and General Embryology and Program in Neuroscience, University of Fribourg, CH-1705, Fribourg, Switzerland.
Brain research (NETHERLANDS) May 2 2000, 864 (1) p142-5, ISSN 0006-8993 Journal Code: 0045503
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Recently identified chondroitin sulphate proteoglycans in perineuronal nets include neurocan and phosphacan. However, the function and assembly of these components has yet to be resolved. In this study we show morphological alteration in Wisteria floribunda labelled nets around cortical interneurones both in tenascin-R knockout and tenascin-R/parvalbumin double knockout mice. This alteration reflects the loss of phosphacan and neurocan from cortical nets in mice deficient in tenascin-R. No effect on the membrane related cytoskeleton, as revealed by ankyrin(R), was observed in any of the mice. These results on mice lacking tenascin-R substantiate previously reported in vitro interactions between tenascin-R and phosphacan and neurocan.
Record Date Created: 20000811
Record Date Completed: 20000811

6/7/18 (Item 13 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12836974 PMID: 10773191
Involvement of chondroitin sulfates on brain-derived tenascin-R in carbohydrate-dependent interactions with fibronectin and tenascin-C.
Probstmeier R; Braunewell K; Pesheva P
Department of Biochemistry, Institute of Animal Anatomy and Physiology, University of Bonn, 53115, Bonn, Germany.
Brain research (NETHERLANDS) Apr 28 2000, 863 (1-2) p42-51, ISSN 0006-8993 Journal Code: 0045503
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Tenascin-R (TN-R), a matrix glycoprotein of the central nervous system (CNS), has been implicated in a variety of cell-matrix interactions involved in the control of axon growth, myelination and cell adhesion to fibronectin during development and regeneration. While most of the functional analyses have concentrated exclusively on the role of the core protein, the contribution of TN-R glycoconjugates present on many potential sites for N- and O-glycosylation is presently unknown. Here we provide evidence that TN-R derived from adult mouse brain expresses chondroitin sulfate (CS) glycosaminoglycans (GAGs), i.e. C-6S and C-4S, that are recognized by the CS/dermatan sulfate-specific monoclonal **antibodies** 473 HD and CS-56. Using ligand-binding, cell adhesion and neurite outgrowth assays, we show that TN-R-linked CS GAGs (i) are involved in the interaction with the heparin-binding sites of fibronectin and are responsible for TN-R-mediated inhibition of cell adhesion to a 33/66-kD heparin-binding fibronectin fragment or to FN-C/H I and FN-C/H II peptides, known to participate in fibronectin binding to cell surface proteoglycans; and (ii) partially contribute to the interaction between TN-R and TN-C which, however, does not lead to an interference with TN-R- and TN-C-mediated inhibition of neurite outgrowth when the two molecules are offered as a mixed substrate in culture. Our findings suggest the functional implication of TN-R-linked CS GAGs in matrix interactions with fibronectin and TN-C that are likely to contribute to a modulation of cellular behavior and the macromolecular organization of matrix components in the developing or injured adult CNS.
Record Date Created: 20000714
Record Date Completed: 20000714

6/7/19 (Item 14 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12793615 PMID: 10723065
Chondroitin sulfates expressed on oligodendrocyte-derived tenascin-R are involved in neural cell recognition. Functional implications during CNS development and regeneration.
Probstmeier R; Stichel C C; Muller H W; Asou H; Pesheva P
Department of Biochemistry, Institute of Animal Anatomy and Physiology, University of Bonn, Bonn, Germany.
Journal of neuroscience research (UNITED STATES) Apr 1 2000, 60 (1) p21-36, ISSN 0360-4012 Journal Code: 7600111
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Tenascin-R (TN-R), an extracellular matrix constituent of the central nervous system (CNS), has been implicated in a variety of cell-matrix interactions underlying axon growth inhibition/guidance, myelination and neural cell migration during development and regeneration. Although most of the functional analyses have concentrated exclusively on the role of the core protein, the contribution of TN-R glycoconjugates present on many potential sites for N- and O-glycosylation is presently unknown. Here we provide first evidence that TN-R derived from whole rat brain or cultured oligodendrocytes expresses chondroitin sulfate (CS) glycosaminoglycans (GAGs), i.e., C-4S and C-6S, that are recognized by CS-56, a CS/dermatan sulfate-specific monoclonal ***antibody*** . Based on different in vitro approaches utilizing substrate-bound glycoprotein, we found that TN-R-linked CS GAGs (1) promote oligodendrocyte migration from white matter microexplants and increase the motility of oligodendrocyte lineage cells; (2) similar to soluble CS GAGs, induce the formation of glial scar-like structures by cultured cerebral astrocytes; and (3) contribute to the antiadhesive properties of TN-R for neuronal cell adhesion in an F3/F11-independent manner, but not to neurite outgrowth inhibition, by mechanism(s) sensitive to chondroitinase or CS-56 treatments. Furthermore, after transection of the postcommissural fornix in adult rat, CS-bearing TN-R was found to be stably upregulated at the lesion site. Our findings suggest the functional impact of TN-R-linked CS on neural cell adhesion and migration during brain morphogenesis and the contribution of TN-R to astroglial scar formation (CS-dependent) and axon growth inhibition (CS-independent), i.e., suppression of axon regeneration after CNS injury.

Record Date Created: 20000412
Record Date Completed: 20000412

6/7/20 (Item 15 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12727730 PMID: 10652443
Tenascin-R inhibits regrowth of optic fibers in vitro and persists in the optic nerve of mice after injury.
Becker T; Anliker B; Becker C G; Taylor J; Schachner M; Meyer R L; Bartsch U
Zentrum fur Molekulare Neurobiologie Hamburg, Universitat Hamburg, Germany. tcbecker@zmnh.uni-hamburg.de
Glia (UNITED STATES) Feb 15 2000, 29 (4) p330-46, ISSN 0894-1491 Journal Code: 8806785
Contract/Grant Number: NS26750; NS; NINDS
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: MEDLINE; Completed

Tenascin-R, an extracellular matrix constituent expressed by oligodendrocytes and some neuronal cell types, may contribute to the inhibition of axonal regeneration in the adult central nervous system. Here we show that outgrowth of embryonic and adult retinal ganglion cell axons from mouse retinal explants is significantly reduced on homogeneous substrates of tenascin-R or a bacterially expressed tenascin-R fragment comprising the epidermal growth factor-like repeats (EGF-L). When both molecules are presented as a sharp substrate border, regrowing adult axons do not cross into the tenascin-R or EGF-L containing territory. All in vitro experiments were done in the presence of laminin, which strongly promotes growth of embryonic and adult retinal axons, suggesting that tenascin-R and EGF-L actively inhibit axonal growth. Contrary to the disappearance of tenascin-R from the regenerating optic nerve of salamanders (Becker et al., J Neurosci 19:813-827, 1999), the molecule remains present in the lesioned optic nerve of adult mice at levels similar to those in unlesioned control nerves for at least 63 days post-lesion (the latest time point investigated), as shown by immunoblot analysis and immunohistochemistry. In situ hybridization analysis revealed an increase in the number of cells expressing tenascin-R mRNA in the lesioned nerve. We conclude that, regardless of the developmental stage, growth of retinal ganglion cell axons is inhibited by tenascin-R and we suggest that the continued expression of the protein after an optic nerve crush may contribute to the failure of adult retinal ganglion cells to regenerate their axons in vivo. Copyright 2000 Wiley-Liss, Inc.

Record Date Created: 20000411
Record Date Completed: 20000411

6/7/21 (Item 16 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12411727 PMID: 9722619

Dissection of complex molecular interactions of neurofascin with axonin-1, F11, and tenascin-R, which promote attachment and neurite formation of tectal cells.

Volkmer H; Zacharias U; Norenberg U; Rathjen F G
Max-Delbruck-Centrum fur Molekulare Medizin, D-13122 Berlin, Germany.

Journal of cell biology (UNITED STATES) Aug 24 1998, 142 (4) p1083-93, ISSN 0021-9525 Journal Code: 0375356
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Neurofascin is a member of the L1 subgroup of the Ig superfamily that promotes axon outgrowth by interactions with neuronal NgCAM-related cell adhesion molecule (NrCAM). We used a combination of cellular binding assays and neurite outgrowth experiments to investigate mechanisms that might modulate the interactions of neurofascin. In addition to NrCAM, we here demonstrate that neurofascin also binds to the extracellular matrix glycoprotein tenascin-R (TN-R) and to the Ig superfamily members axonin-1 and F11. Isoforms of neurofascin that are generated by alternative splicing show different preferences in ligand binding. While interactions of neurofascin with F11 are only slightly modulated, binding to axonin-1 and TN-R is strongly regulated by alternatively spliced stretches located in the NH2-terminal half, and by the proline-alanine-threonine-rich segment. In vitro neurite outgrowth and cell attachment assays on a neurofascin-Fc substrate reveal a shift of cellular receptor usage from NrCAM to axonin-1, F11, and at least one additional protein in the presence of TN-R, presumably due to competition of the neurofascin- NrCAM interaction. Thereby, F11 binds to TN-R of the neurofascin/TN-R complex, but not to neurofascin, whereas axonin-1 is not able to bind directly to the neurofascin/TN-R complex as shown by competition binding assays. In conclusion, these investigations indicate that the molecular interactions

of neurofascin are regulated at different levels, including alternative splicing and by the presence of interacting proteins.
Record Date Created: 19980925
Record Date Completed: 19980925

6/7/22 (Item 17 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12387168 PMID: 9698315
Tenascin-R is antiadhesive for activated microglia that induce downregulation of the protein after peripheral nerve injury: a new role in neuronal protection.
Angelov D N; Walther M; Streppel M; Guntinas-Lichius O; Neiss W F; Probstmeier R; Pesheva P
Department of Anatomy, University of Cologne, 50924 Cologne, Germany.
Journal of neuroscience - the official journal of the Society for Neuroscience (UNITED STATES) Aug 15 1998, 18 (16) p6218-29, ISSN 0270-6474 Journal Code: 8102140
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Microglial activation in response to pathological stimuli is characterized by increased migratory activity and potential cytotoxic action on injured neurons during later stages of neurodegeneration. The initial molecular changes in the CNS favoring neuronofugal migration of microglia remain, however, largely unknown. We report that the extracellular matrix protein tenascin-R (TN-R) present in the intact CNS is antiadhesive for activated microglia, and its downregulation after facial nerve axotomy may account for the loss of motoneuron protection and subsequent neurodegeneration. Studies on the protein expression in the facial and hypoglossal nucleus in rats demonstrate that TN-R is a constituent of the perineuronal net of motoneurons and 7 d after peripheral nerve injury becomes downregulated in the corresponding motor nucleus. This downregulation is reversible under regenerative (nerve suture) conditions and irreversible under degenerative (nerve resection) conditions. In short-term adhesion assays, the unlesioned side of brainstem cryosections from unilaterally operated animals is nonpermissive for activated microglia, and this nonpermissiveness is almost abolished by a monoclonal ***antibody*** to TN-R. Microglia-conditioned media and tumor necrosis factor-alpha downregulate TN-R protein and mRNA synthesis by cultured oligodendrocytes, which are one of the sources for TN-R in the brainstem. Our findings suggest a new role for TN-R in neuronal protection against activated microglia and the participation of the latter in perineuronal net destruction, e.g., downregulation of TN-R.
Record Date Created: 19980909
Record Date Completed: 19980909

6/7/23 (Item 18 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12279697 PMID: 9589384
Defasciculation of neurites is mediated by tenascin-R and its neuronal receptor F3/11.
Xiao Z C; Revest J M; Laeng P; Rougon G; Schachner M; Montag D
Department of Neurobiology, Swiss Federal Institute of Technology, ETH-Hoenggerberg, Zurich, Switzerland.
Journal of neuroscience research (UNITED STATES) May 15 1998, 52 (4) p390-404, ISSN 0360-4012 Journal Code: 7600111
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: MEDLINE; Completed

Fasciculation and defasciculation of axons are major morphogenetic events in the formation of neuronal pathways during development. We have identified the extracellular matrix glycoprotein tenascin-R (TN-R) and its neuronal receptor, the immunoglobulin superfamily recognition molecule F3, as promoters of neurite defasciculation in cerebellar explant cultures. Perturbation of the interaction between these two molecules using both **antibodies** and an antisense oligonucleotide resulted in increased neurite fasciculation. The domains involved in defasciculation were identified as the N-terminal region of TN-R containing the cysteine-rich stretch and the 4.5 epidermal growth factor-like repeats and the immunoglobulin-like domains of F3. Fasciculation induced by **antibodies** and the antisense oligonucleotide could be reverted by a phorbol ester activator of protein kinase C, whereas the protein kinase inhibitor staurosporine increased fasciculation. Our observations indicate that defasciculated neurite outgrowth does not only depend on the reduction of the expression of fasciculation enhancing adhesion molecules, such as L1 and the neural cell adhesion molecule (NCAM), but also on recognition molecules that actively induce defasciculation by triggering second messenger systems.

Record Date Created: 19980715
Record Date Completed: 19980715

6/7/24 (Item 19 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12132733 PMID: 9433801

The distribution of tenascin-R in the developing avian nervous system.

Derr L B; McKae L A; Tucker R P

Department of Neurobiology and Anatomy, Wake Forest University School of Medicine, Winston-Salem, North Carolina 27157-1010, USA.

Journal of experimental zoology (UNITED STATES) Feb 1 1998, 280 (2) p152-64, ISSN 0022-104X Journal Code: 0375365

Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Tenascin-R is an extracellular matrix glycoprotein found in the developing central nervous system. In vitro, tenascin-R promotes axon growth. In order to learn more about the function and regulation of tenascin-R in neural development, we have used monoclonal **antibodies** to localize tenascin-R immunoreactivity in the developing and adult chicken brain. The earliest stage stained with anti-tenascin-R was embryonic day 6.5, when the matrix surrounding the cell bodies of spinal cord motoneurons was stained. At embryonic day 18, specific brain regions were stained, including the basal nuclei, habenular nuclei, layer "i" of the optic tectum, and the motor nuclei of cranial nerves. Anti-tenascin-R staining persisted in these regions in the adult chicken brain. Thus, the distribution of tenascin-R immunoreactivity in the developing and mature avian brain is correlated with the distribution of nerve growth factor (NGF)-sensitive neurons, but it is not correlated with neurite outgrowth. Finally, NGF was shown to upregulate tenascin-R expression in PC12 cells, indicating that expression of neuron-derived tenascin-R may be regulated by exogenous NGF.

Record Date Created: 19980204
Record Date Completed: 19980204

6/7/25 (Item 20 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11892941 PMID: 9169525

Tenascin-R is an intrinsic autocrine factor for oligodendrocyte differentiation and promotes cell adhesion by a sulfatide-mediated mechanism.

Pesheva P; Gloor S; Schachner M; Probstmeier R

Department of Physiology, Neurophysiology, Institute of Animal Anatomy and Physiology, University of Bonn, Bonn, Germany.

Journal of neuroscience - the official journal of the Society for Neuroscience (UNITED STATES) Jun 15 1997, 17 (12) p4642-51, ISSN 0270-6474 Journal Code: 8102140

Publishing Model Print; Erratum in J Neurosci 1997 Aug 1;17(15) 6021

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

O4(+) oligodendrocyte (OL) progenitors in the mammalian CNS are committed fully to terminal differentiation into myelin-forming cells. In the absence of other cell types in vitro, OL differentiation reproduces the in vivo development with a correct timing, suggesting the existence of an intrinsic regulatory mechanism that presently is unknown. We have examined the effect of two isoforms of the extracellular matrix (ECM) molecule tenascin-R (TN-R), which is expressed by OLs during the process of myelination, on the adhesion and maturation of OLs in vitro. Here we show that the substrate-bound molecules supported the adhesion of O4(+) OLs independently of the CNS region or age from which they were derived. At the molecular level this process was mediated by protein binding to membrane surface sulfatides (Sulf), as indicated by the interference of O4 **antibody** and Sulf with the attachment of OLs or other Sulf+ cells, erythrocytes, to TN-R substrates and by direct protein-glycolipid binding studies. In the absence of platelet-derived growth factor (PDGF), exogenous TN-R induced myelin gene expression and the upregulation of its own synthesis by cultured cells, resulting in a rapid terminal differentiation of O4(+) progenitors. Our findings strongly suggest that TN-R represents an intrinsic regulatory molecule that controls the timed OL differentiation by an autocrine mechanism and imply the relevance of TN-R for CNS myelination and remyelination.

Record Date Created: 19970630

Record Date Completed: 19970630

6/7/26 (Item 21 from file: 155)

11175758 PMID: 7479846

The versican C-type lectin domain recognizes the adhesion protein tenascin-R.

Aspberg A; Binkert C; Ruoslahti E

Cancer Research Center, La Jolla Cancer Research Foundation, CA 92037, USA.

Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Nov 7 1995, 92 (23) p10590-4, ISSN 0027-8424 Journal Code: 7505876

Contract/Grant Number: CA28896; CA; NCI; CA30199; CA; NCI; CA42507; CA; NCI

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

The core proteins of large chondroitin sulfate proteoglycans contain a C-type lectin domain. The lectin domain of one of these proteoglycans, versican, was expressed as a recombinant 15-kDa protein and shown to bind to insolubilized fucose and GlcNAc. The lectin domain showed strong binding in a gel blotting assay to a glycoprotein doublet in rat brain extracts. The binding was calcium dependent and abolished by chemical deglycosylation treatment of the ligand glycoprotein. The versican-binding glycoprotein was identified as the cell adhesion protein tenascin-R, and versican and tenascin-R were both found to be localized in the granular layer of rat

cerebellum. These results show that the versican lectin domain is a binding domain with a highly targeted specificity. It may allow versican to assemble complexes containing proteoglycan, an adhesion protein, and hyaluronan.
Record Date Created: 19951221
Record Date Completed: 19951221

6/7/27 (Item 22 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11040932 PMID: 7615642
Characterization of functional domains of the tenascin-R (restrictin) polypeptide: cell attachment site, binding with F11, and enhancement of F11-mediated neurite outgrowth by tenascin-R.
Norenberg U; Hubert M; Brummendorf T; Tarnok A; Rathjen F G
Max-Delbruck-Centrum fur Molekulare Medizin, Berlin, Germany.
Journal of cell biology (UNITED STATES) Jul 1995, 130 (2) p473-84, ISSN 0021-9525 Journal Code: 0375356
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The extracellular matrix glycoprotein tenascin-R (TN-R) is a multidomain protein implicated in neural cell adhesion. To analyze the structure-function relationship of the different domains of TN-R, several recombinant TN-R fragments were expressed in bacterial cells. Two distinct binding regions were localized on the TN-R polypeptide: a region binding the axon-associated immunoglobulin (Ig)-like F11 protein and a cell attachment site. The binding region of the glycosylphosphatidylinositol (GPI)-anchored F11 was allocated to the second and third fibronectin type III (FNIII)-like domain within TN-R. By using a mutant polypeptide of F11 containing only Ig-like domains, a direct interaction between the Ig-like domains of F11 and FNIII-like domains 2-3 of TN-R was demonstrated. The interaction of TN-R with F11 in in vitro cultures enhanced F11-mediated neurite outgrowth, suggesting that the combined action of F11 and TN-R might be of regulatory influence on axon extension. A cell attachment region was identified in the FNIII-like domain eight of TN-R by domain-specific ***antibodies*** and fusion constructs. This site is distinct from the F11 binding site within TN-R.
Record Date Created: 19950822
Record Date Completed: 19950822

6/7/28 (Item 23 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10832812 PMID: 7530144
Tenascin glycoproteins in developing neural tissues: only decoration?
Faissner A; Scholze A; Gotz B
Department of Neurobiology, University of Heidelberg, Germany.
Perspectives on developmental neurobiology (UNITED STATES) 1994, 2 (1) p53-66, ISSN 1064-0517 Journal Code: 9417971
Publishing Model Print
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Tenascin glycoproteins constitute a growing family of extracellular matrix molecules that are transiently expressed by astrocytes during the development of the central nervous system (CNS). In some areas, tenascin distribution is discrete and tenascin boundaries delineate emerging functional processing units, for example, in the somatosensory cortex. The intact adult CNS displays low levels of expression and up-regulation of

tenascin after lesion or in glial tumors. In vitro studies using the purified glycoprotein, bacterially expressed tenascin fusion proteins, monoclonal **antibodies** , and defined cell culture models have established that tenascin glycoproteins possess cell-binding sites for neural cells, support neuronal migration and the formation of neurites. On the other hand, tenascin also embodies repulsive, inhibitory properties that could serve to conceal neuronal assemblies and to segregate emerging fiber tracts. These functional properties are encoded by distinct domains of the molecule and suggest that tenascin glycoproteins are involved in neural pattern formation and regeneration. This interpretation is discussed with reference to a recent report that the elimination of the tenascin gene does not cause obvious abnormalities of neural tissues. (118 Refs.)
Record Date Created: 19950222
Record Date Completed: 19950222

6/7/29 (Item 24 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10418475 PMID: 8294147
Astrocytes and neurons regulate the expression of the neural recognition molecule ***janusin*** by cultured oligodendrocytes.
Jung M; Pesheva P; Schachner M; Trotter J
Department of Neurobiology, University of Heidelberg, Germany.
Glia (UNITED STATES) Nov 1993, 9 (3) p163-75, ISSN 0894-1491
Journal Code: 8806785
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Janusin (formerly designated J1-160/180) is an extracellular matrix glycoprotein highly homologous to tenascin, consisting of two major molecular forms of 160 and 180 kD expressed by oligodendrocytes and in myelin. ***Janusin*** expression is upregulated during myelination and in the adult it remains expressed at lower levels. It is also present at the node of Ranvier, where myelin, axon, and astrocytic process are in close contact. To gain an understanding of the regulatory mechanisms which may underlie expression of **janusin**, the differentiation stage-dependent expression of **janusin** was studied in cultures enriched in mouse oligodendrocytes and their precursor cells. Expression of ***janusin*** by these cells was highest on both A2B5+ and O4+/O1- oligodendroglial precursor cells and a subset of myelin associated glycoprotein-positive (MAG+) oligodendrocytes. Hardly any of the more differentiated O1+ or O10+ oligodendrocytes expressed ***janusin*** . Expression of ***janusin*** was influenced by co-culture with astrocytes or neurons. Astrocytes or astrocytic-conditioned culture supernatants elevated the expression of **janusin** by the more differentiated oligodendrocytes (O1+ or MAG+ cells), while its expression by oligodendroglial precursor cells was relatively unchanged. Platelet-derived growth factor, but not basic fibroblast growth factor, also elevated the expression of **janusin** by O1+ or O10+ oligodendrocytes. In contrast, co-culture with neurons originating from dorsal root ganglia or spinal cord decreased the expression of cell-bound **janusin** by oligodendrocytes and their precursor cells. These observations indicate that expression of **janusin** on these cells in culture is susceptible to opposing regulatory influences from astrocytes and neurons. Such influences may modulate the temporal and spatial distribution of **janusin** in the developing and adult central nervous system.
Record Date Created: 19940228
Record Date Completed: 19940228

6/7/30 (Item 25 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10366632 PMID: 8244531
Expression of **janusin** (J1-160/180) in the retina and optic nerve of the developing and adult mouse.
Bartsch U; Pesheva P; Raff M; Schachner M
Department of Neurobiology, Swiss Federal Institute of Technology, Zurich.
Glia (UNITED STATES) Sep 1993, 9 (1) p57-69, ISSN 0894-1491 Journal Code: 8806785
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
We have analyzed the expression of the oligodendrocyte-derived extra-cellular matrix molecule **janusin** (previously termed J1-160/180) in the retina and optic nerve of developing and adult mice using indirect light and electron microscopic immunocytochemistry, immunoblot analysis, and enzyme-linked immunosorbent assay. In the optic nerve, ***janusin*** is not detectable in neonatal and only weakly detectable in 7-day-old animals. Expression is at a peak in 2- or 3-week-old animals and subsequently decreases with increasing age. In the retina, expression increases until the third postnatal week and then remains at a constant level. In immunocytochemical investigations at the light microscopic level, **janusin** was found in the myelinated regions of the nerve with spots of increased immunoreactivity possibly corresponding to an accumulation of the molecule at the nodes of Ranvier. At the electron microscopic level, contact sites between unmyelinated axons, between axons and glial cells, and between axons and processes of myelinating oligodendrocytes were immunoreactive. Cell surfaces of astrocytes at the periphery of the nerve and forming the glial-limiting membrane, in contrast, were only weakly immunopositive or negative. In cell cultures of young postnatal mouse or rat optic nerves, oligodendrocytes and type-2 astrocytes, but not type-1 astrocytes were stained by ***janusin*** ***antibodies*** . In the oligodendrocyte-free retina, **janusin** was detectable in association with neuronal cell surfaces, but not with cell surfaces of Muller cells or retinal astrocytes. Our observations indicate that expression of **janusin** in the optic nerve and in the retina is developmentally differentially regulated and that other cell types, in addition to oligodendrocytes, express the molecule. Since the time course of **janusin** expression in the optic nerve coincides with the appearance of oligodendrocytes and myelin and since **janusin** is associated with cell surfaces of oligodendrocytes and outer aspects of myelin sheaths and is concentrated at nodes of Ranvier, we suggest that **janusin** is functionally involved in the process of myelination.
Record Date Created: 19931229
Record Date Completed: 19931229

6/7/31 (Item 26 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10066585 PMID: 7678967
The F3/11 cell adhesion molecule mediates the repulsion of neurons by the extracellular matrix glycoprotein J1-160/180.
Pesheva P; Gennarini G; Goridis C; Schachner M
Department of Neurobiology, Swiss Federal Institute of Technology, Zurich.
Neuron (UNITED STATES) Jan 1993, 10 (1) p69-82, ISSN 0896-6273 Journal Code: 8809320
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The oligodendrocyte-derived extracellular matrix protein J1-160/180

displays repellent substrate properties toward neurons. In a search for neuronal ligands mediating the response to J1-160/180, we have identified the F3/11 cell surface protein, a glyco-phosphatidylinositol-anchored member of the immunoglobulin superfamily. F3/11 mediates the initial recognition between a J1-160/180 substrate and cerebellar neurons or F3-transfected CHO cells. In cerebellar neurons, the F3/11-J1-160/180 interaction induces a repulsion consisting of the loss of substrate adhesion with time in culture and inhibition of neurite outgrowth. **Antibody** blocking experiments show that the avoidance response of neurites at J1-160/180 substrate borders is also mediated by F3/11. Active cell-cell and cell-substrate repulsion is considered a major mechanism governing the extent and directionality of axonal growth, but the ligand-receptor interactions involved have remained unknown. Our results show that F3/11 mediates the neuronal response to the repellent molecule J1-160/180 and may thus be involved in signal transduction leading to cell repulsion.
Record Date Created: 19930310
Record Date Completed: 19930310

6/7/32 (Item 27 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09537024 PMID: 1717703
Identification of a cDNA clone specific for the oligodendrocyte-derived repulsive extracellular matrix molecule J1-160/180.
Fuss B; Pott U; Fischer P; Schwab M E; Schachner M
Department of Neurobiology, Swiss Federal Institute of Technology, Honggerberg.
Journal of neuroscience research (UNITED STATES) Jul 1991, 29 (3) p299-307, ISSN 0360-4012 Journal Code: 7600111
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
A cDNA clone specific for the oligodendrocyte-derived extracellular matrix glycoproteins J1-160/180 was obtained from a lambda ZAPII expression library using polyclonal ***antibodies*** generated against mouse J1-160. The library was constructed from poly(A)(+)-RNA isolated from O1 antigen-positive rat oligodendrocytes. The cDNA clone expressed a fusion protein that was recognized by the J1-160/180-specific monoclonal ***antibodies*** 596, 619, and 620, and, weakly, 597. The fusion protein was not recognized by polyclonal ***antibodies*** to mouse J1/tenascin. The cDNA clone with an insert of approximately 5.6 kb in size contained the nucleotide sequence coding for the amino acid sequence of the N-terminus of a tryptic peptide derived from mouse J1-160. The developmental and tissue distribution of the mRNA recognized by the cDNA clone is in agreement with the described expression of the J1-160/180 proteins.
Record Date Created: 19911104
Record Date Completed: 19911104

6/7/33 (Item 28 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09186826 PMID: 1701406
Expression of the adhesion molecules L1, N-CAM and J1/tenascin during development of the murine small intestine.
Probstmeier R; Martini R; Tacke R; Schachner M
Department of Neurobiology, University of Heidelberg, Federal Republic of Germany.
Differentiation; research in biological diversity (GERMANY) Jul 1990, 44 (1) p42-55, ISSN 0301-4681 Journal Code: 0401650
Publishing Model Print

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

We have previously studied the immunohistological localization of the three adhesion molecules L1, N-CAM and J1/tenascin in adult mouse small intestine and shown that L1 expression in epithelial crypt cells underlies the adhesion of these cells to one another [63]. To obtain further insight into the functional roles of L1, N-CAM and J1/tenascin in this organ we studied their expression starting at embryonic day 14 during embryonic and early postnatal morphogenesis and during epithelial cell migration in the adult. Expression of L1 was restricted to neural cells until approximately postnatal day 5, when L1 started to be detectable on crypt but not on villus cells, predominantly on the basolateral membrane infoldings. As in brain, L1-specific mRNA was approximately 6 kb in size. L1 from intestine appears to differ from the brain-derived equivalent in possessing a higher level of glycosylation. N-CAM was detectable from embryonic day 14 onward in neural and also in mesenchymal cells. Expression by smooth muscle cells decreased during development. In the villus core, N-CAM was strongly detectable at contact sites between smooth muscle cells forming the cellular scaffold of the villus. From embryonic day 14 onward, N-CAM appeared in both 180- and 140-kDa forms. J1/tenascin was present in both neural and mesenchymal cells from embryonic day 14 onward. Starting at embryonic day 17, J1/tenascin appeared concentrated at the boundary between mesenchyme and epithelium in an increasing gradient from the crypt base to the villus top. From embryonic day 14 onward J1/tenascin consisted of the 190- and 220-kDa components. J1/tenascin from intestine differed from brain-derived J1 in its carbohydrate composition. These observations show that the three adhesion molecules are expressed by distinct cell populations and may serve as cell-type-specific markers in pathologically altered intestinal tissue.

Record Date Created: 19910124
Record Date Completed: 19910124

6/7/34 (Item 29 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08748775 PMID: 2477380

J1-160 and J1-180 are oligodendrocyte-secreted nonpermissive substrates for cell adhesion.

Pesheva P; Spiess E; Schachner M
Department of Neurobiology, University of Heidelberg, Federal Republic of Germany.

Journal of cell biology (UNITED STATES) Oct 1989, 109 (4 Pt 1) p1765-78, ISSN 0021-9525 Journal Code: 0375356
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The glia-derived J1 extracellular matrix glycoproteins have been referred to as J1-160/J1-180 (the developmentally late appearing lower molecular weight group) and J1-200/J1-220 (the developmentally early appearing higher molecular group immunochemically related to tenascin). Members of the two groups show distinct cross-reactivities. To characterize the structural and functional differences between these J1 glycoproteins, two monoclonal **antibodies** were generated which recognize only the members of the lower molecular weight group. The two ***antibodies*** detect immunochemical similarities among the members of the lower molecular weight group, but do not react with J1/tenascin. J1-160 and J1-180 are specifically expressed by differentiated oligodendrocytes in culture and by myelin of the central nervous system and have not been found in the peripheral nervous system nor in any other organ of the adult mice tested. Electron microscopic examination of rotary-shadowed J1-160 and J1-180 reveals, respectively, dimeric and trimeric (tribrachion) kink-armed

rodlike structures, which are linked by disulfide bridges. J1-160/J1-180 are nonpermissive substrates for the attachment and spreading of early postnatal small cerebellar neurons, astrocytes, and fibroblasts. In a mixture with laminin, J1-160/J1-180 are nonpermissive substrates for neurons, but not for astrocytes or fibroblasts. The repulsive effect toward neurons can be neutralized by one of the monoclonal **antibodies**, but not by the other. These observations are discussed in the context of cell interactions during regeneration in the mammalian nervous system.
Record Date Created: 19891102
Record Date Completed: 19891102

6/7/35 (Item 30 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08314390 PMID: 2456237
The high-molecular-weight J1 glycoproteins are immunochemically related to tenascin.
Faissner A; Kruse J; Chiquet-Ehrismann R; Mackie E
Department of Neurobiology, Heidelberg, Federal Republic of Germany.
Differentiation; research in biological diversity (GERMANY) 1988, 37 (2) p104-14, ISSN 0301-4681 Journal Code: 0401650
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The J1 glycoproteins have been shown to mediate neuron-astrocyte adhesion and appear in the nervous system as four species of Mr 160,000 (J1-160), 180,000 (J1-180), 200,000 (J1-200), and 220,000 (J1-220), respectively. Tenascin is a disulfide-linked oligomeric, extracellular matrix glycoprotein of subunit Mr 170,000, 190,000, 200,000, and 220,000, which has been proposed to promote epithelial cell proliferation. In view of the structural similarities of the molecules we have used immunohistochemical and immunochemical techniques to compare them. Immunohistochemically, polyclonal J1 and tenascin **antibodies** yielded identical staining patterns in non-nervous-system tissues, and staining could be completely blocked by preincubating the sera with purified tenascin. In the central nervous system all structures expressing tenascin immunoreactivity were also recognized by J1 ***antibodies*** . However, not all J1-positive structures were also tenascin-positive, indicating that J1 **antibodies** recognized additional epitopes not present on tenascin. Western-blot experiments performed with affinity-purified polyclonal J1 **antibodies** showed that J1 glycoproteins can be subdivided into two separate pairs, J1-160/180 and J1-200/220, which share a small degree of homology. Western-blot experiments and sequential immunoprecipitations on biosynthetically [35S]methionine- or 125I-radiolabeled J1 glycoproteins carried out with polyclonal J1 and tenascin **antibodies** demonstrated that J1-200/220 is immunochemically indistinguishable from tenascin. These observations suggest that one set of extracellular glycoproteins is associated with processes as different as neural histogenesis and carcinogenesis of mammary glands.
Record Date Created: 19880830
Record Date Completed: 19880830

6/7/36 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

136259601 CA: 136(17)259601p PATENT
Highly sensitive proteomic analysis methods and kits and systems for practicing the same
INVENTOR(AUTHOR): Tchaga, Grigoriy S.
LOCATION: USA
ASSIGNEE: Clontech Laboratories Inc.

PATENT: PCT International ; WO 200225288 A2 DATE: 20020328
APPLICATION: WO 2001US29658 (20010921) *US PV234527 (20000922)
PAGES: 89 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: G01N-033/68A; B01J-019/00B DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; PH; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ ; SD; SL; SZ; TZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA209016 Biochemical Methods
IDENTIFIERS: proteome array immunoassay immobilization antibody epitope binding pectin kit
DESCRIPTORS:
Integrins...
α5; highly sensitive proteomic anal. methods and kits and systems for practicing same
Apparatus...
array; highly sensitive proteomic anal. methods and kits and systems for practicing same
Sulfones...
as linking group; highly sensitive proteomic anal. methods and kits and systems for practicing same
Proteins...
Bax; highly sensitive proteomic anal. methods and kits and systems for practicing same
Catenins...
β-; highly sensitive proteomic anal. methods and kits and systems for practicing same
Cyclins...
CDC27; highly sensitive proteomic anal. methods and kits and systems for practicing same
Proteoglycans,analysis...
chondroitin sulfate-containing, neuroglycan C; highly sensitive proteomic anal. methods and kits and systems for practicing same
Proteins...
dematin; highly sensitive proteomic anal. methods and kits and systems for practicing same
Distillation columns...
fractional; highly sensitive proteomic anal. methods and kits and systems for practicing same
Glass,analysis... Calretinin... Test kits... Epitopes...
Polysaccharides,analysis... Proteins... Fluorescent substances... Labels...
Apple... Extraction... Sample preparation... Buffers... pH... Washing...
Chelating agents... Ions... Fractionation... Affinity chromatography...
Fluorometers... Immobilization,molecular... Immunoassay... Proteome...
highly sensitive proteomic anal. methods and kits and systems for practicing same
Glycoproteins...
Janusin; highly sensitive proteomic anal. methods and kits and systems for practicing same
Glutamate receptors...
metabotropic, mGluR1; highly sensitive proteomic anal. methods and kits and systems for practicing same
Proteins...
microtubule-associated, MAP 4 (microtubule-associated protein 4); highly sensitive proteomic anal. methods and kits and systems for practicing same
Proteins...
microtubule-associated, MAP2B; highly sensitive proteomic anal. methods and kits and systems for practicing same
Antibodies...
monoclonal; highly sensitive proteomic anal. methods and kits and systems for practicing same

Cell adhesion molecules...
N-CAM, receptor; highly sensitive proteomic anal. methods and kits and systems for practicing same
Proteins...
PSD95; highly sensitive proteomic anal. methods and kits and systems for practicing same
Microscopes...
slides; highly sensitive proteomic anal. methods and kits and systems for practicing same
CAS REGISTRY NUMBERS:
9000-69-5 102623-58-5 9046-38-2 4044-65-9 141467-21-2 208939-71-3
highly sensitive proteomic anal. methods and kits and systems for practicing same

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            9085  JANUS
         1764100  BOD?
      S8       0  JANUS(W)BOD?
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ANSWER 1 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:303330 CAPLUS
DN 131:110261
ED Entered STN: 18 May 1999
TI Molecular architecture with metal ions, nucleobases and other heterocycles
AU Navarro, Jorge A. R.; Lippert, Bernhard
CS Fachbereich Chemie, Universitat Dortmund, Dortmund, 44221, Germany
SO Coordination Chemistry Reviews (1999), 185-186, 653-667
CODEN: CCHRAM; ISSN: 0010-8545
PB Elsevier Science S.A.
DT Journal; General Review
LA English
CC 78-0 (Inorganic Chemicals and Reactions)
AB A **review**, with over 40 refs., is given on transition metal amine complexes with nucleobases. The combination of transition metal fragments with 90 or **180**.**degree**. **angles**, viz. cis-a2MII (a = amine or a2 = diamine; M = Pt or Pd) and trans-a2PtII or HgII, resp., with heterocyclic ligands providing 90, 120, or **180** .**degree**. **angles** leads, either in a self-assembly process or in a stepwise fashion, to cations of versatile shapes and charges. Examples include open boxes, mol. squares, hexagons, triangles and cups. Intermol. H bonding can assist association of smaller entities. Synthetic and structural aspects as well as physicochem. properties of selected examples are discussed.
ST **review** transition metal amine nucleobase prepn structure
IT Transition metal complexes
Transition metal complexes
RL: PRP (Properties); SPN (Synthetic preparation); PREP (Preparation)
(amine; preparation and mol. architectures of palladium, platinum and mercury amine complexes with nucleobases)
IT Hydrogen bond
(in palladium, platinum and mercury amine complexes with nucleobases)
IT Molecular structure
Self-assembly
Supramolecular structure
(preparation and mol. architectures of palladium, platinum and mercury amine complexes with nucleobases)
IT Amines, preparation
Amines, preparation
Nucleic acid bases
RL: PRP (Properties); SPN (Synthetic preparation); PREP (Preparation)
(transition metal complexes; preparation and mol. architectures of palladium, platinum and mercury amine complexes with nucleobases)
IT 7439-97-6DP, Mercury, nucleobase complexes, preparation 7440-05-3DP, Palladium, nucleobase complexes, preparation 7440-06-4DP, Platinum, nucleobase complexes, preparation
RL: PRP (Properties); SPN (Synthetic preparation); PREP (Preparation)
(preparation and mol. architectures of palladium, platinum and mercury amine complexes with nucleobases)
RE.CNT 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD

ANSWER 1 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:303330 CAPLUS
DN 131:110261
ED Entered STN: 18 May 1999
TI Molecular architecture with metal ions, nucleobases and other heterocycles
AU Navarro, Jorge A. R.; Lippert, Bernhard
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CODEN: CCHRAM; ISSN: 0010-8545
PB Elsevier Science S.A.
DT Journal; General Review
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CC 78-0 (Inorganic Chemicals and Reactions)
AB A **review**, with over 40 refs., is given on transition metal amine complexes with nucleobases. The combination of transition metal fragments with 90 or **180**.**degree**. **angles**, viz. cis-a2MII (a = amine or a2 = diamine; M = Pt or Pd) and trans-a2PtII or HgII, resp., with heterocyclic ligands providing 90, 120, or **180** .**degree**. **angles** leads, either in a self-assembly process or in a stepwise fashion, to cations of versatile shapes and charges. Examples include open boxes, mol. squares, hexagons, triangles and cups. Intermol. H bonding can assist association of smaller entities. Synthetic and structural aspects as well as physicochem. properties of selected examples are discussed.
ST **review** transition metal amine nucleobase prepn structure
IT Transition metal complexes
Transition metal complexes
RL: PRP (Properties); SPN (Synthetic preparation); PREP (Preparation)
(amine; preparation and mol. architectures of palladium, platinum and mercury amine complexes with nucleobases)
IT Hydrogen bond
(in palladium, platinum and mercury amine complexes with nucleobases)
IT Molecular structure
Self-assembly
Supramolecular structure
(preparation and mol. architectures of palladium, platinum and mercury amine complexes with nucleobases)
IT Amines, preparation
Amines, preparation
Nucleic acid bases
RL: PRP (Properties); SPN (Synthetic preparation); PREP (Preparation)
(transition metal complexes; preparation and mol. architectures of palladium, platinum and mercury amine complexes with nucleobases)
IT 7439-97-6DP, Mercury, nucleobase complexes, preparation 7440-05-3DP, Palladium, nucleobase complexes, preparation 7440-06-4DP, Platinum, nucleobase complexes, preparation
RL: PRP (Properties); SPN (Synthetic preparation); PREP (Preparation)
(preparation and mol. architectures of palladium, platinum and mercury amine complexes with nucleobases)
RE.CNT 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD